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ARCHIVES OF PEDIATRICS

A MONTHLY DEVOTED TO THE

DISEASES OF INFANTS AND CHILDREN

JOHN FITCH LANDON, M.D., Editor

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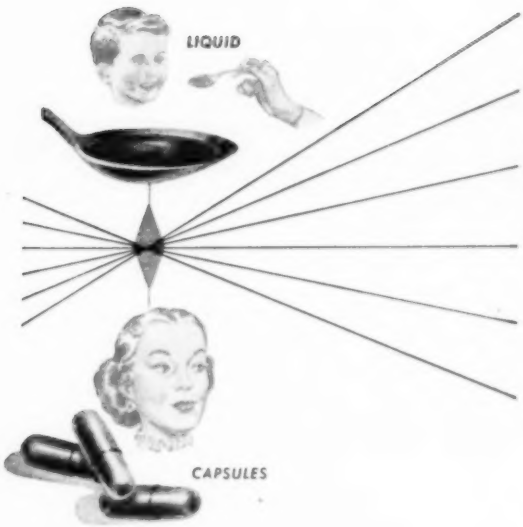

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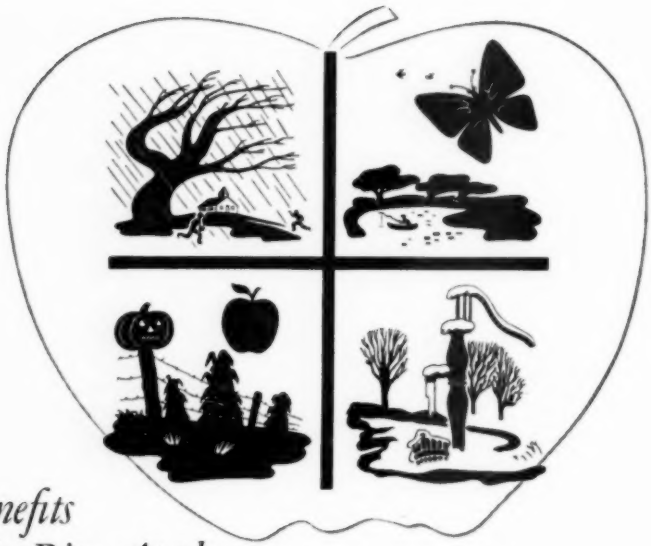

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THE TRUTH ABOUT
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In view of the above findings, more and more physicians now specify Minute Maid Fresh-Frozen Orange Juice in lieu of home-squeezed orange juice where optimum year-around intake of natural Vitamin C is indicated.

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Wallace R. Roy, Ph.D., Director of Research

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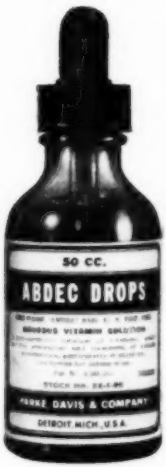

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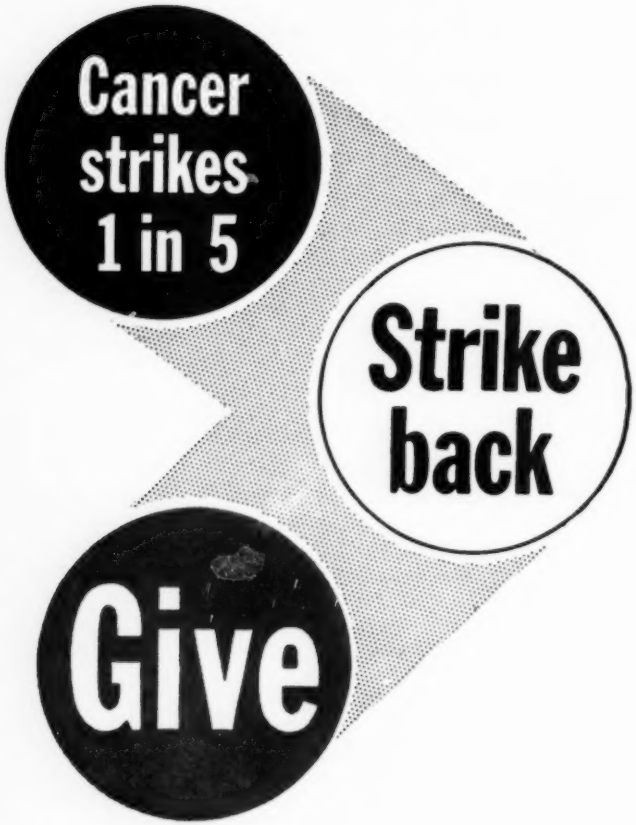

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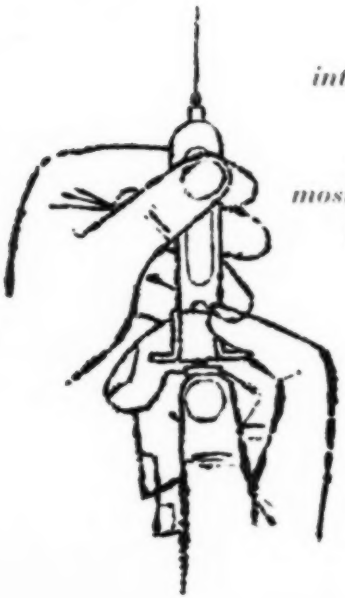

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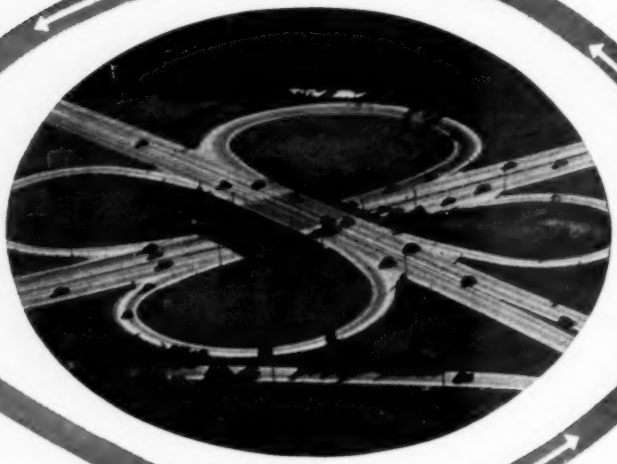

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OBSTRUCTIVE LARYNGEAL LESIONS IN THE INFANT*

JOHN J. O'KEEFE, M.D.
Philadelphia.

Having survived the first few days of life without incident, a certain number of infants, sooner or later, are seen because of evidencing the symptoms of laryngeal obtruction. The seriousness of such situations varies from the insidious and slowly developing obstruction due to new growth to the rapidly progressive dyspnea of foreign body lodgement. In many of these instances the determination of the cause of obstruction is a trying and perplexing diagnostic problem.

That such situations are seldom easily or quickly solved is evidenced in the increasing tendency for those engaged in the practice of pediatrics to consult with the endoscopist in the management of these patients. Their joint efforts, even when coupled with the assistance of an experienced roentgenologist, are sometimes taxed to the extreme before obtaining the solution of a particular issue. Rarely may the symptoms of laryngeal obstruction, no matter in what combination of sequence of occurrence, be relied upon to indicate the nature or cause of that obstruction. The evolving of such symptoms is solely an index of laryngeal malfunction, and to be most accurately interpreted must be gauged according to their correllary functions as normally appreciated.

Briefly summarized, and in the order of their importance, the functions of the larynx, and the symptoms produced by disruption of those functions are:

*From the Department of Laryngology and Broncho-Esophagology, Jefferson Hospital, Philadelphia.

1. *Respiration.* Except in foreign body accidents and in developmental anomalies, changes in respiratory function, due to laryngeal obstruction are ordinarily slow to occur, and may amount to little more than a slight increase in respiratory rate. This rate increases directly with the degree or amount of obstruction, and as objective dyspnea develops with progression of obstruction, breathing becomes audible, and ordinarily is referred to as wheezing or stridorous respiration. The wheeze or stridor of laryngeal dyspnea typically is more pronounced during the inspiratory phase of respiration, although in extreme dyspnea it may be heard also during expiration. In contrast, foreign body lodgement, commonly resulting in sudden dyspnea and made worse as secondary edema and inflammatory reaction of the traumatized mucosa develops, somewhat mimics the stridor of congenital malformations, which also characteristically is a persistant one and is recognized as such soon after birth.

2. *Phonation.* "Hoarseness"—or any other term used to designate abnormal sound production—is the most common single sympton of laryngeal disease, and most commonly occurs first in this sequence of symptoms. This is apparent when it is recalled that the integrity of mobility and approximation of the vocal cords is affected by many disorders that do not at all impinge on the glottic space. As applied to infants with laryngeal obstruction, change in the character of the crying sound must serve as the criterion of "hoarseness."

3. *Protection.* The cough-reflex is the most poorly understood and the least appreciated function of the larynx. This is especially so in dealing with the infant wherein accidents of tracheobronchial aspiration happen so easily and so often with depression of laryngeal sensation. Excessive coughing, dry or nonproductive, is commonly observed as a consequence of excessive local irritation, either mechanical or inflammatory.

CLASSIFICATION AND CHACTERISTICS OF LARYNGEAL LESIONS

The purpose of this preliminary discussion of the elements of laryngeal physiology is to show and to emphasize that a symptomatic diagnosis amounts to no diagnosis; and that in order to institute proper and effective therapy an etiologic or causative diagnosis must be made. This is more clearly appreciated in a cor-

sideration of the specific laryngeal disorders effecting obstruction.

1. *Developmental Anomalies.* Contained within this grouping are several entities, all evidenced at birth or soon thereafter, and all characterized principally by the symptom of stridor. Commonly encountered is the situation of excessive weakness of the larynx, laryngomalacia, in which it has been made possible for the soft tissues to be drawn into the glottis during each inspiratory effort. Varying degrees of this deformity are observed, most of them compatible with life, but occasionally one of extreme development is encountered necessitating operative intervention. A somewhat similar picture is created wherein the infantile epiglottis is found to be unnaturally flaccid and possible loosely fixed by means of its glosso-epiglottic ligaments, allowing for its posterior displacement into the glottis. Web formation, another congenital anomaly, occurs unusually and, again, in varying degrees of involvment, and if not diagnosed promptly implies serious consequence to the future health and possible life of the infant.

Fig. 1.

"Infantile" epiglottis with posterior displacement. Congenital web stenosis.

Symptomatically, one cannot differentiate these deformities. There are no distinguishing marks to the symptom of stridor, regardless of its degree of severity, that single out one deformity from the others. And in view of the fact that one web formation is corrected only by surgery, the others as diagnostic possibilities must be excluded. Direct laryngoscopic examination is the only way by which such a differential diagnosis can be made. To defer this examination, or to assume the possibilty of spontaneous correction taking place, is an unwarranted and unjustifiable assump-

tion and one that unnecessarily places the child's life in jeopardy.

2. *Acute Infections.* Probably the most common single cause of laryngeal obstruction in infants is that due to the inflammatory reaction of acute upper respiratory tract infections. This is particularly apparent during the course of epidemic respiratory tract disease. Ordinarily, while there is some degree of laryngeal involvment in the sporatic type of upper respiratory tract infection, there appears to be a predilection for obstructive laryngeal reaction in the epidemic forms of this disease. In such instances the sequence of events is sometimes so rapid in development that an emergency situation exists before prophylactic or effective symptomatic therapy can be instituted. It behooves one, then, to be ever aware of the possibility of such obstruction occurring during these

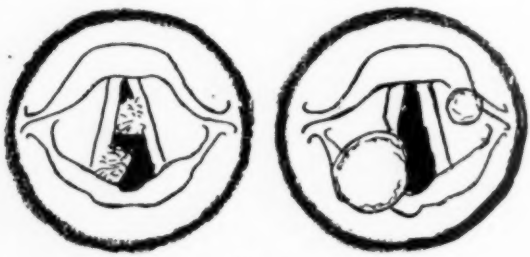

Fig. 2.

Infantile papilloma. Laryngeal cysts.

epidemics, and to prepare to cope with such situations on short notice. The composite clinical picture of acute laryngotracheobronchitis in itself is sufficiently typical as to amount to a diagnosis —at least sufficiently so to indicate the need for measures intended to maintain adequate laryngeal respiratory function.

A singular reaction to upper respiratory tract infection is occasionally noted wherein the epiglottis is principally involved. In such instances stridor will be noted to develop without the forewarning of hoarseness.

3. *Cysts and Tumors.* In a large clinic, papillomata are relatively frequently encountered as a cause of laryngeal obstruction in infants. Occurring in such an age group these tumorous masses are probably truly neoplastic, and characteristically occur as mutiple papillomata involving any part, or combination of parts, of the

larynx, even to extending into the trachea. Such masses, having origin on the true cords, are very apt to be a cause of respiratory embarrassment. Dyspnea, in this instance, is usually slow in developing, although progression frequently advances to an alarming degree. Other types of solid tumors rarely occur in the infant larynx. Cysts of varying size have been recorded as occurring on the ventricular bands and on the aryepiglottic folds, and, depending on their size and position, may very effectively produce laryngeal obstruction. Other than being congenital, these, at times, are of the retention type.

Here again are a related group of causative factors capable of producing laryngeal obstruction in an infant. Identification cannot be made, based alone on the clinical characteristics presented by any one of them. Again, the differential diagnosis is dependent upon direct laryngoscopic inspection.

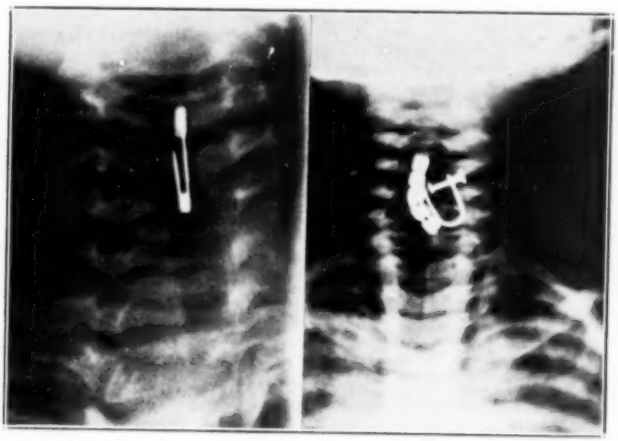

Fig. 3.

Foreign body—open safety pin—lodged in the larynx. Foreign body—earring—lodged in the laryngopharynx.

4. *Trauma.* The mobility of the larynx, being dependent upon the integrity of the recurrent nerves, is occasionally disrupted in instances of birth trauma, either by the direct action of instrumental delivery or, at times, by the effects of traction; rare instances are reported in which these paralyses have been caused by difficult

and prolonged labors precipitating intracranial changes. Fortunately, most such accidents result only in unilateral paralyses. And fortunately, too, recovery of function frequently occurs spontaneously. Unilateral paralyses of the larynx seldom result in obstruction of sufficient extent as to cause actual objective dyspnea in a newborn infant. Bilateral paralyses, in which the vocal cords assume a median position, leave little glottic space. The stridor in such instances is severe and demanding of immediate relief.

A similar type of trauma is occasionally responsible for dislocation of the arytenoid cartilages. Such dislocations are usually toward the midline and, although the respiratory space is reduced, the reduction is not a critical one.

5. *Foreign Body.* The accidental aspiration and lodgement of foreign objects in the larynx produce a situation of immediate seriousness. Initially, while the degree of obstruction is dependent upon the size and/or shape of the foreign body, the secondarily developing inflammatory reaction caused by this traumatic accident seriously furthers the degree of obstruction. In an instance of suspected foreign body aspiration, it is well to remember to have the neck, as well as the chest, included in the roentgen ray study; truly a technical factor, but one which is occasionally seen to be a point of embarrassment to the physician, and one of more critical embarrassment to the patient. It is axiomatic, too, that any patient suspected of having aspirated a foreign body, and in whom symptoms of respiratory distress have been or are present, even in spite of a negative roentgen-ray study, should have the advantages of endoscopic examination before finally being labelled negative.

Occasionally, when large or odd shaped foreign bodies find lodgement behind the larynx, they may exert sufficient pressure on the soft and yielding cartilaginous structures of the infant's larynx as to cause severe stridor. This again illustrates the importance of a well-done roentgen study; in cases of non-opaque foreign bodies it may be necessary to enlist the aid of contrast media in order to define the presence of such objects.

COMMENT

By way of enumerating the common characterisics of specific lesions causing laryngeal obstruction in infants, it is illustrated that reliance cannot be placed on the symptoms of laryngeal ob-

struction as a means of attaining a causative diagnosis. Many morbid conditions of the larynx produce situations that symptomatically have a great deal in common. Granted there are at times factors that help individualize such instances and do lend a sense of probability to the making of such diagnoses, they are so unusual and so variable in occurrence that they cannot be engaged as dependable sources through which proper judgement may be formulated.

The differential diagnosis of almost all such disorders is made only by means of direct laryngoscopy. This procedure in the hands of a competent and trained endoscopist is performed with the least degree of trauma. This same procedure is likewise utilized as the means to specific therapy in many such instances.

Acute obstructive laryngeal dyspnea in the infant, which has progressed to the point of respiratory embarrassment, commonly characterized by stridor, indrawing of the soft tissues, and restlessness of the patient, should be considered a threatening to life. In such instances, and especially so in those in which the progression of symptoms of obstruction is rapid, the life saving virtue of tracheotomy may be the first and only therapeutic procedure worth considering. Recourse to this type of surgical intervention should not be deferred until cyanosis or the insidious effects of anoxia are noted, it being better to perform this procedure a little too soon rather than a little too late. The detrimental consequences of tracheotomy are of minor importance when compared to its beneficial effects.

255 *South 17th Street.*

FAMILIAL OCCURRENCE OF ANKYLOSING SPONDYLITIS. (Nederlandsch Tijdschrift v. Geneeskunde, Amsterdam, 95:3763, Dec. 15, 1951). The 296 patients with ankylosing spondylitis (Bechterew's disease) reviewed by de Blécourt belonged to 239 families; 96 cases occurred in 39 families and the remaining 200 cases were the only ones in a family. In two families there were four cases, and in one family five cases. Another interesting observation in this group was that 70 of 296 patients, or nearly one-fourth, were women. This percentage is considerably higher than the average of 10 per cent usually given in the literature.—*Journal A.M.A.*

DOMESTIC WATER FLUORIDATION

Its Pathogenic Potentialities

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Toronto, Canada

For many years the dental condition known as "mottled teeth", or "Texas teeth", has been recognized in both mankind and in domestic animals in many parts of the American Southwest; but not until the research of Professors H. V. and Margaret Smith,[1] biochemists of the University of Arizona, in 1930, was it discovered that fluorine in the natural waters of these regions was the culpable agent. For a decade, following this discovery, public health researchers and industrial engineers sought by physical or chemical means to devise methods for the removal of fluorine or mitigation of its toxic effects in domestic water supplies. A summary of such methods may be found in the April 1935 number of the American Journal of Public Health.

Shortly before the last world war, when Hereford, a town in Deaf Smith county, Texas, was heralded as the "Town without a Toothache," a new angle was projected into the problem. The unusually good condition of the teeth of children in this locality was studied by dentists and public health workers, and the assumption that the natural fluorine content of the local water supply was the beneficent agent was quickly made. It was noted that even in the cases where the teeth were mottled the incidence of decay was apparently minimized. In seeking the cause of this anomaly little attention was given to the possible favorable effects of sunlight in producing natural vitamin D, or to the possible superior mineral content of the foods grown in that region, or to the fact that these same regions were prolific in production of citrus fruit with its high content of vitamin C, although all these factors may have contributed.

Such an alternative explanation is supported by the work of Dr. R. S. Harris, Director of the Nutritional Biochemistry Laboratories of the Massachusetts Institute of Technology. He fed two groups of hamsters milk and corn from Texas and New England. The animals receiving the Texas food developed only half as much tooth decay as those fed the New England food, although the amount of fluorine in the Texas food was so little different from that of the New England food that it could not appreciably affect the result of the experiment. Dr. Harris also found that riboflavin,

a non-toxic B-vitamin nutrient, was almost as effective as fluorine in preventing caries.

Further evidence of the role of nutrition in dental health is provided by the work of Dr. M. T. Hanke and an associate group of physicians and dentists,[2] who, in 1933, conducted an unusual experiment in the town of Mooseheart, Illinois. In this community, privately owned by the Order of the Moose, some 1300 orphans were housed and cared for by a competent staff of teachers, dieticians, recreational directors, etc. For purpose of the experiment some 200 of these children were given a daily dietary supplement of twenty ounces of orange juice, while the remainder, who were fed otherwise in the same manner on a good average diet, were used as controls. The experiment was continued for three years, during which time the dental and general health status of all the children was carefully charted. It was found that the group receiving the extra citrus-fruit supplement had a very much lower incidence of new dental cavities and a marked freedom from gingivitis and pyorrhea, as well as a greatly lowered incidence of colds and acute infectious diseases, as compared to the control group. It was thus shown that malnutrition, with special emphasis on vitamin C deficiency, provides the background for bad dental health and the usual infectious diseases of childhood. On this basis dental caries should not be considered a separate disease entity, but rather as an index of nutritional status and constitutional resistance to infectious disease.

The precipitous conclusion regarding the relation of fluorine to dental health has quickly led to agitation for mass application of artificial sodium fluoride to domestic water supplies in an effort to duplicate the supposed favorable effect of the fluorine found in natural waters, and already some twenty million freedom-loving Americans are compelled to partake of this medicated beverage irrespective of its possible long-term deleterious effects.

The historical background of fluorine may provide related interest. Fluorine was discovered by Scheele in 1771, but because of its intense affinities for so many other elements it was not produced in gaseous elemental purity until 1886 by Moissan. Because of its most active affinity it has been regarded as the wild cat or villain of the chemical world, being also the most poisonous. The inhalation of the pure gas is invariably lethal. The soluble forms, as in sodium fluoride, are extremely poisonous, being used commercially for rat and roach extermination. The nonsoluble forms,

as in calcium fluoride, are much less toxic. Sodium fluoride is the form proposed for general use in domestic water supplies. It is an artificial chemical, specially prepared from mineral fluor spar for use in the production of aluminum. Otherwise, heretofore it has been a well-nigh useless product, having limited commercial use because of its extreme toxicity. Among its few industrial uses are the following: vermin extermination, laundry bleach, rubber coagulant, fixer in dye works, flux in metallurgy, glass etching, optical grinding paste, glue manufacture, emery wheel binder and concrete hardener. Its value in the last two items is due to its strong affinity for calcium—the same property which makes it unite with the calcium of teeth and bones.

The geographical source of fluorine is related to volcanic activity. For this reason it is found in highest concentration in the southwestern mountainous regions of New Mexico and Arizona. There are many extinct volcanoes in these parts. The spread of fluorine eastward extends only as far as the Alleghenies. The continued ingestion of vegetable and animal food from these areas has resulted in the cumulative deposition of this element in the bones and teeth of man and animals in combination with the calcium of these structures. For this reason fluorine should not be considered a biological requisite. The bones of cattle on the western prairies have been found to be so hard and stony from high fluorine content that the U. S. Department of Agriculture has forbidden the use of such bones in the production of bone meal for human consumption except from animals under 2½ years of age. Dr. E. V. McCollum, the great nutritionist, says fluorine serves no useful purpose in the animal organism.

It is the strong affinity of fluorine for calcium which results in its deposition in dental and osseous tissues, and which, when present below a certain critical level, builds into the calcifying dental enamel an increased density and hardness which gives apparent resistance to dental decay. This is essentially a chemical reaction, analogous to the industrial use of fluorine in the hardening of cement and concrete. It is nevertheless a pathogenic process, as is also the deposition of calculus (tartar) on the teeth, which also is known to give a certain degree of immunity to caries, while at the same time it is conducive to periodontal disease—gingivitis and pyorrhea. Fluorine likewise may be concurrently conducive to periodontal disease. Dr. H. K. Box,[3] a periodontist of international repute, on the Dental Faculty of the University of Toronto, in-

forms the writer that he has checked the dental status of a number of subjects who have been using naturally fluoridated water for several decades and has found appalling periodontal disease, with marked morphological changes, such as enlargement of the roots and narrowing or closure of root canals and pulp chambers, indicating premature aging of the tooth structure, making extraction very difficult and prone to root fracture. Many of these cases had severe gingivitis and pyorrhea and premature need for dentures.

When the long continued ingestion of a foreign substance (fluorine) results in such marked local changes in the teeth, it is only logical to assume the possibility of remote systemic effects. Such findings have been reported by a group of Danish researchers among workers in cryolite mining who were inhaling fluorine in dust. Not only did these subjects exhibit the above-mentioned dental defects (thickened roots and narrowed pulp chambers), but supercalcification was also manifested in osteophyte formations on bones, osteosclerosis, calcification of tendons, "poker backs" and fixation of thoracic wall. These same effects have been noted also in parts of Africa and India where the natives have long been using naturally fluoridated water. Associated malnutrition seemed to accentuate the defects.

In England, in 1948, bone defects in three groups of schoolboys were studied. Two groups were from districts where the water supply was practically free from fluorine. The third group was from the town of Launton, where the natural water supply contains 1 part per million of fluorine. X-ray examination revealed that 20 per cent of the first two groups had mild nonspecific spinal irregularities. In the third group, 64 per cent were found to have spinal defects and the lesions were more severe.

Brandl and Tappeiner[4] fed very small amounts of sodium fluoride to young growing dogs for 21 months and examined their bones microscopically. Deposited in the bones were found vivid crystals of calcium fluoride. They believed that soluble fluorine salts combine with the calcium of the bones, causing increased brittleness. The bones of fluorine-fed animals are always chalky and fragile as are the teeth.

Recently, in Switzerland, cattle grazing in the vicinity of aluminum smelters were found to be suffering from a fatal disease which veterinarians diagnosed as fluorosis, a form of osteomalacia producing spontaneous fracturing of bones. The cattle had acquired

the disease from eating the grass upon which were deposited fluorine emanations from the aluminum plant.

More recently (1952), Udall and Keller[5] have reported an outbreak of fluorosis in cattle on farms about five miles distant from an aluminum plant near Vancouver, Wash. The animals were subjected to increased intake of fluorine from the fumes of smoke

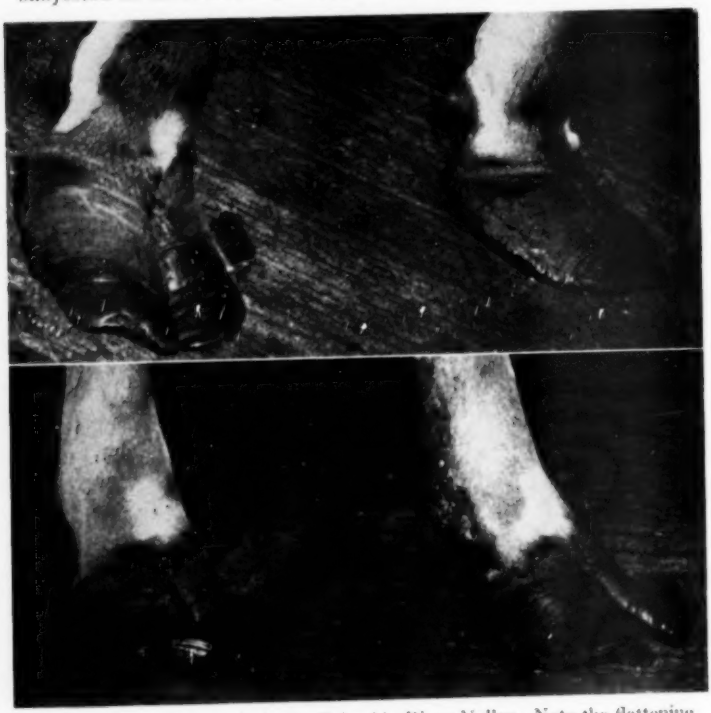

Fig. 1. Fluorosis in cattle in the Columbia River Valley. Note the flattening and distortion of hooves.

stacks. In rainy or foggy weather the toxic effects were greatly increased, due to moisture taking up the fluorine and depositing same on the cattle fodder. The younger animals seemed to be affected mostly in stunted growth and marked disturbances in reproduction, lactation and alimentation. The leg joints were noticeably enlarged, inhibiting locomotion. The hooves were soft and spreading, with grotesque distortions. The teeth were increasingly blackened. These lesions were obviously due to extreme deple-

tion of calcium, the latter being taken up from the food in the alimentary tract to form the insoluble calcium fluoride, thus preventing assimilation. The alimentary and reproductive disturbances were related to the toxic action of the fluorine. A surplus of calcium in the diet apparently compensates for the fluorine-calcium loss and to some extent saves bone deficiences, but it has little effect in retarding tooth damage, owing perhaps to the more direct exposure of the teeth to the fluorine intake in addition to the internal systemic effect. This may explain why children of low-income families, with minimal milk intake, are found to be more sensitive to fluorine.

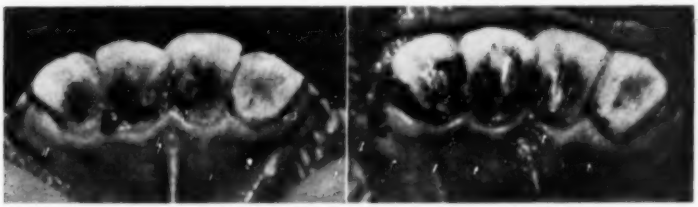

Fig. 2. Fluorosis in cattle in the Columbia River Valley. Note the mottling of teeth and the progression in three months' interval.

The Smiths,[1] of Arizona, previously referred to as co-discoverers of fluorine as a cause of dental mottling, report studies in the naturally fluoridated areas indicating that while the incidence of caries was low in young children the degree of decay increased sharply after 21 years of age (or after 21 years exposure to fluorine), and furthermore that the decay and weakening of tooth structure was so severe that repair was more difficult, resulting in a high percentage of extractions. Even in communities, where the fluorine content of water is only 1 part to 1,000,000, they report fluorosis (mottling) in 10 to 12 per cent of the inhabitants.

Regarding the effect of fluorine on body enzymes: Sodium fluoride is known to be particularly harmful to the action of lipase in fat metabolism. Solutions with a fluorine content as low as 1 part in 15,000,000 may inhibit the activity of lipase as much as 50 per cent. The enzymatic breakdown of carbohydrates is also very sensitive to fluorine. It has been found that the normal catabolic transition to lactic acid and pyruvic acid is greatly disturbed, thus nullifying to some extent the catalytic action of thiamine.

Regarding the effect of fluorine on blood coagulation: Stuber and Lang[6] report a high blood level of fluorine in a number of

cases of hemophilia. There seemed to be a direct correlation between the blood-fluorine level and the prolongation of the blood coagulation time. Knowing that goose blood and rabbit blood clot slowly, they found on investigation a high blood-fluorine content in these animals. Cat and dog blood, which are known to clot rapidly, was found to be free from fluorine. (This last finding may be due to the fact that carnivorous animals eat bones, the calcium of which would take up the fluorine in the alimentary tract and prevent absorption—W.J.M.) Continuing their observations they found that human subjects, using drinking water with varying content of natural fluorine, had a blood coagulation time 6 to 20 times that of subjects using fluorine-free water.

Smith and Leverton[7] report studies regarding the relative toxicity of various fluorides in the experimental feeding of rats. They found, for instance, that sodium fluoride is 85 times more toxic than calcium fluoride, as determined by lethal dosage. The obvious reason for this disparity is the fact that sodium fluoride is very soluble, whereas calcium fluoride is practically insoluble. (Sodium fluoride is the form used in artificial fluoridation, while calcium fluoride is found in natural waters.

Carnot,[8] a French scientist, who studied the flourine content of bones, was the first to suggest a method for removing fluorine from drinking water (1893) by treating it with pulverized bone or bone ash. By this means the fluorine was taken up by the calcium of bones outside the body before it could gain access to the bones within the body.

From the foregoing it must be evident that domestic water fluoridation at best is a poor substitute for better nutrition. When major dependence is placed on such measures, public interest in food reform and oral hygiene is sure to suffer.

In conclusion I wish to offer suggestions, based on laboratory experiments, for protection against the toxic effects of fluorine for those who find themselves unwilling subjects of this latest experiment in mass medication.

1. The daily ingestion of small doses (5 to 10 grains) of certain innocuous salts of calcium, notably calcium lactate, citrate, gluconate or ascorbate, which should provide a surplus of calcium for reaction with the sodium fluoride of the drinking water, thereby producing an insoluble precipitate (calcium fluoride) which is then mostly eliminated without absorption from the alimentary tract. Calcium ascorbate is particularly valuable in this respect, since

it at the same time releases vitamin C, the antitoxic action of which should help to counteract the toxic effect of that portion of the sodium fluoride which may be absorbed. Vitamin C has been used successfully as an antidote in lead poisoning, also for rattlesnake and scorpion venom, and it should be equally effective in counteracting rat poison.

2. An optimal intake of foods rich in calcium is desirable, such as milk products and green salad vegetables. A surplus of these in the diet provides the calcium necessary for neutralizing the fluoride as previously explained. Bone meal, as a dietary supplement, should have similar effect.

3. Last, but not least, make sure of an optimal intake of citrus fruits to provide the natural vitamin C to help counteract the toxic fluoride.

SUMMARY

The literature regarding fluorosis in men and animals is reviewed, with special reference to dental and systemic effects.

The writer admits the apparent immunity to dental caries conferred in young children by either topical application or ingestion of fluorine in dilution of one part or less per million, but regards the local dental reaction as a pathogenic process analogous to the deposition of calculus (tartar), resulting in severe periodontal disease in later life, as well as possible deleterious systemic effects.

Suggestions regarding prophylactic medicinal and dietary measures to counter the toxic effect of fluorine in drinking water are offered for those who find themselves unwilling subjects of experimentation.

ACKNOWLEDGMENT: The author wishes to thank Dr. D. H. Udall, Professor Emeritus, Cornell University, for the loan of the cuts re "Fluorosis in Cattle" as used in this paper.

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16 *Gothic Avenue.*

CLINICAL REVIEW

In order to encourage the writing of clinical articles by recent graduates or senior medical students, the ARCHIVES *will publish monthly at least one such paper from the classes of Doctor Reuel A. Benson, New York Medical College, New York, and Doctor Philip Moen Stimson, Cornell Medical School, New York. Other interested medical schools are cordially invited to submit student papers for consideration.*

ETIOLOGY AND OCCURRENCE OF ARTERIAL HYPERTENSION IN POLIOMYELITIS*

A REVIEW OF THE LITERATURE

ELIZABETH CORYLLOS, M.D.
New York.

There are at present several prevailing ideas about the cause and perpetuation of arterial hypertension in poliomyelitis. One is that it is primarily neurogenic in its origin and perpetuation; another that it is primarily respiratory in origin, being secondary to hypoxia (either increased carbon dioxide tension in the blood, decreased oxygen or both). A third idea, and possibly the most correct, is that it can be due to either of these factors, and in the great majority of cases to a combination of both. This seems to be borne out by the fact that there is an increase in the incidence of hypertension with increasing severity of the disease. Its greatest incidence is in those cases where bulbar and respiratory symptoms are the most evident. The neurogenic theory receives support from the known high incidence of virus invasion of the medulla as seen at autopsy. The respiratory or anoxia theory is supported by the high incidence of hypertension and its longer duration in all cases with respiratory embarrassment, especially in those that have required respirators for any length of time.

Baker[26] and his associates have shown that cardiovascular signs and symptoms are consistently associated with severe damage to more than 19 per cent of the large reticular cells of the medulla. In the experience of Plum and Wolff[5], arrythmias and shock, due to failure of neurogenic cardiovascular regulation, proved unresponsive to therapeutic measures, such as administration of digitalis glycosides, neosynephrin and parenteral fluids.

*This paper was awarded first prize by Dr. Philip Moen Stimson for papers submitted in a competition by students of Cornell University Medical College, New York City.

In contrast to the discouraging prognosis offered by circulatory failure, the ill effects of respiratory insufficiency can often be prevented or reversed, and therefore the maintenance of a physiologic respiratory exchange is given primary attention, especially in these bulbar cases. The importance of avoiding hypoxia and the dangers of the hypoxic state have in general received greater emphasis than have the incidence and sequelae of carbon dioxide retention. Hypoxia results in increased capillary permeability, pulmonary edema, cardiovascular collapse and damage to the central nervous system. High levels of carbon dioxide in the blood (hypercapnia) result from inadequate alveolar elimination of the gas, and, if not reduced, lead to acidosis, headache, hypertension[6], disorientation and loss of consciousness. Hypercapnia may also depress the respiratory center and cause vascular collapse. Nearly all these effects of hypoxia or hypercapnia may further interfere with respiratory gas exchange to intensify the severity of an acute illness. The prevention or reversal of even borderline respiratory decompensation is thus of first importance in managing patients with acute poliomyelitis. Deficiencies in ventilation are due to paralysis of the diaphragm or intercostal muscles, depression or destruction of the respiratory center in the medulla, or obstruction of the upper airway resulting from pharyngeal or laryngeal paralysis. Alveolar deficiency may be the result of pulmonary edema, atelectasis or pneumonia.

Arterial hypertension was a striking finding in over half of the acute paralytic polio patients seen at New York Hospital during 1949 and 1950[18]. It appeared in the acute stage of the disease and sometimes lasted well into convalescence. Its degree was generally in proportion to the severity of the illness. The observations made at this time suggest that hypertension is a relatively common associated finding in acute polio, and may occur in patients with with comparatively benign forms of the disease. This is also borne out by the small series of cases presented by Vickers[15] in 1940. This was composed of a group of young adults between 20 and 30 who were in good health except for an elevated blood pressure, some as high as 180/120. The only positive past history in the members of this group was an attack of anterior poliomyelitis during childhood. The great majority of this group had little or no residual paralysis or atrophy of muscle groups.

In the New York Hospital group, hypertension was considered to be present if a patient maintained a diastolic blood pressure of over 90 for a period of more than twelve hours. A history of hypertension was sought in all cases, but was negative in all but one. Hypertension, by these criteria, was found in 45 of 95 adolescents and adults with acute anterior poliomyelitis. The normal expected incidence in a similar normal group would be less than 9.1 per cent. Males were affected twice as often as females. The highest incidence of hypertension occurred in patients suffering bulbospinal paralysis or paralysis of all extremities. Many of this group needed artificial respiration. In the entire series, only one patient died who had not had hypertension.

Hypertension was of considerably greater degree and longer duration in patients needing artificial respiration than it was in non-respirator patients. In the hypertensive respirator patients who survived the acute illness, the average blood pressure was 164/104 and the average duration of hypertension greater than 98 days. In the hypertensive non-respirator patients the average blood pressure was 143/96 and lasted an average of 5.5 days. Only one respirator patient failed to develop a hypertension lasting more than 21 days. The part played by anoxemia or high blood carbon dioxide concentrations in the production of hypertension in these patients was difficult to assess; as was also whether or not the respirator, per se, had an adverse effect on the circulation.

Although frequent plasma carbon-dioxide-combining-power and arterial-oxygen-saturation studies failed to reveal hypercapnia or hypoxemia in many of these patients, it is evident that any patient with acute polio requiring artificial respiration may suffer at least brief undetected periods of anoxemia or hypercapnia. Such short periods of hypoxemia or hypercapnia may have served to intensify the hypertension in these patients. Many of them, however, developed their hypertension hours before there was any detectable decrease in their vital capacity as measured by the spirometer. Also, brief periods of hypoxemia or hypercapnia would not be expected to produce prolonged hypertension. In these respirator patients, hypoxemia and hypercapnia appeared to be neither universally present nor the most probable specific causes of their hypertension. The patients in this series who were treated with the rapid-rocking bed developed a hypertension as great and as

protracted as that of most of the patients treated with tank respirators.

Three patients with the Guillain-Barré syndrome who were treated in New York Hospital during this same period also were given artificial respiration with tank respirators for periods of five days to three weeks. Each of these patients developed hypertension ranging from 160/100 to 230/160 while in the respirator. But, in contrast to the polio patients, their blood pressure returned promptly to normal when they no longer needed the respirators and were taken out of them.

The hypertension in the polio patients was found to be a relatively uncomplicated state. Transient papilledema[17] was found during early convalescence in three of the respirator patients who had had hypertension, and also in two other non-respirator cases who did not have hypertension. Abnormalities in the optic fundi in polio are apparently rare and have been noted by only a few of those who have written on poliomyelitis. These patients were recovering from polio with moderate to severe quardiplegia. Papilledema was first observed 12 to 48 days after the acute onset of the illness and persisted from 13 to 140 (average 60) days. Common complaints leading to this observation were diplopia with associated blurred vision. The spinal fluid pressures were elevated. The clinical course in each of these cases was marked by headaches, fever lasting five to 10 days or more, and the development of muscular paralysis with a typical anterior horn cell distribution. No patient exhibited any defects in sensation, and pleocytosis in the spinal fluid was found at the time of onset of the illness. Painful stiff muscles were present in each patient as his disease progressed, and each patient still had significant muscle wasting and weakness nine to 20 months after the onset of the acute attack.

The ophthalmological findings of these patients were indistinguishable from those seen in optic neuritis. However, presence of normal visual acuity, and the increased spinal fluid pressures appeared to indicate that the changes in the fundi were the result of increased intracranial pressure, and hence represented true papilledema.

Since two of the patients with papilledema did not have elevated blood pressure it was thought that the hypertension was not the cause of the papilledema. Appearing mainly in the convalescent

stage, it was benign, and a self-limited complication, perhaps reflecting generalized post-infectious edema of the central nervous system, entirely independent of any hypertension. Of all the patients who had hypertension for over three months, only one had any detectable abnormality in any of the kidney function tests.

In the patients in this series who died, it was found at autopsy that there was significant involvement of the hypothalamus and medullary reticular substance. These areas contain autonomic nuclei that have vasopressor effects and cause arterial hypertension when stimulated. All but one of the patients in whom this was demonstrated had had hypertension. In the patients who survived, and who had clinical evidence of bulbospinal or severe upper-spinal cord involvement, adjacent invasion of these brain-stem autonomic structures could be inferred, and it was in this group that the highest incidence of hypertension occurred.

Bodian[12] has demonstrated that damage to the autonomic centers of the brain may occur in any patient with polio, whether the disease be mild or severe. This suggests that hypertension may have been the result of central-nervous-system changes even in patients with paralysis confined to the lower extremities.

Another factor in the etiology of the hypertension which cannot be ignored is the part which reaction to an alarming situation may produce. Hypertension, as a part of the "alarm reaction," is well documented, and polio is certainly a disease with terrifying potential consequences. The period of adjustment to a respirator is associated with great fear, and it was in such patients that the most marked blood pressure elevations were seen.

The temporary hypertension occurring in these patients may have been initiated by inflammation of the central-nervous-system, or may have been part of a generalized body reaction to a prolonged alarm state. The potentially harmful circulatory effects resulting from the use of the respirator, or the occurrence of transient hypoxemia and hypercapnia appeared to be all that was necessary to convert a transient hypertensive state into a seemingly permanent one. Vicker's[15] finding of hypertension in 11 of 44 young adults who had had polio many years previously suggests that the non-respirator group may also carry an increased susceptibility to the future development of "essential" hypertension

Renal hypertensive mechanisms may have been stimulated to produce an irreversible hypertensive state.

In this connection the blood of five of the polio patients and one of the Guillain-Barré syndrome cases was examined for the presence of the renal vasoexcitor material (VEM)[16], by Lee in Shorr's laboratory. In all of the patients tested significant VEM titers were found. The titers were exceptionally high in three of the polio patients and in the Guillain-Barré patient. All four had hypertension and had received artificial respiration. VEM titers were much lower in the two polio patients who were not hypertensive.

It has been found[19] that age does not make any marked difference as to the incidence of hypertension in acute polio. In Steigman's[19] group of polio patients seen during 1950 he found that out of 32 non-paralytic polio patients there were four with hypertension, or approximately 12 per cent. His criteria for hypertension was that the mean arterial pressure be in excess of 15mm. mercury above normal for that given age, and that it be present on more than three days. Of 140 patients with paralysis, but without respiratory involvement, there were 50 who had hypertension, or approximately twenty-eight per cent.

He was also able to study a group of about 112 children who had had polio in 1949 or before, and found none that were still hypertensive. This may or may not be interpreted as conflicting with Vickers' findings. Steigman also found that dehydrogenated ergot alkaloids had only a mild effect in reducing this hypertension. Tetra-ethyl-ammonium ion and proto-veratrin also lack effect. He also points out that in other neurological diseases, hypertension, constant or intermittent, may occur in paraplegics. Paraplegics, following spinal injury are known to have periodic crises during which very elevated blood pressures are seen, which seem to respond to TEA. During the vascular crises of tabes there is also hypertension. Elevated blood pressures are also seen in post-diphtheritic paralysis.

Sarnoff and Whittenberger[19 (1) and 9] have pointed out that while acutely ill bulbar polio patients with hypertension were put on electrophrenic respiration there would be a very sudden drop in blood pressure to normal levels, and as soon as the machine was turned off the hypertension would return. This was done with

great reproducibility, but was not, however, observed in patients who had had their disease more than one month.

Cardiac output studies performed on a 14-year-old boy, with polio quadriplegia, by injection of the dye T1824 by a No. 6 catheter into the right atrium were not in harmony with a pure neurogenic hypertension, such as may be induced in the experimental animal.

Whittenberger found the respiratory effect on blood pressure to be in proportion to the change in mean intrapleural pressure. The greatest deleterious effect was with intermittent positive pressure. An intermediate effect was observed with alternating positive and negative pressures; this has about the same effect on venous return as normal breathing. A third grade of response was obtained by the enhancement of venous return when the mean intrapleural pressure was more negative than usual, as obtained by vigorous use of phrenic stimulation. If one produced a normal amount of ventilation with phrenic stimulation, the effect on the venous return would be the same as in normal breathing.

Hodes[19(1)] suggested, as possible further evidence of the neurogenic origin of hypertension, and also of the increased secretions in the pharynx in polio, the syndrome described by Conrad Riley of New York. Riley has observed a number of children who are unable to produce tears when crying; they have hypertension and drooling, sometimes actually choking with the secretions. They have flushing and blushing. Some of them have vomiting when disturbed or upset. One of these children was found to have a hypothalamic cyst, which was confirmed at autopsy. The assumption has therefore been made that the other children in this group have lesions of a congenital nature in the hypothalamic region as well as in other areas of the brain. He felt that in polio patients lesions in the hypothalamic area might account for some of the effects seen, such as elevated blood pressure, etc.

Dexter points out that in hypertension in polio, in contrast to renal and essential hypertension, very few vascular complications have been observed (eye ground changes, hemorrhages, exudates, narrow vessels, cardiac and renal lesions). He suggests that in those patients who do develop these complications the sequence of events may be: stone formation, leading to pyelonephritis, and then to hypertensive and vascular complications. In the experimental animal where neurogenic hypertension can be produced by

resection of the carotid and aortic sinuses, there is a very striking relationship between pulse rate and blood pressure, and no vascular complications arose from this neurogenically induced hypertension.

Grulee and Panos[31] describe the symptoms and clinical management in a series of 464 cases of acute polio which they had studied, in which 23 per cent of all cases were of the bulbar type. One hundred per cent of the deaths occurred in this group, and of the ones who recovered, almost all of them recovered completely with no residua. Central cardiorespiratory failure was considered to be responsible for all but one of the deaths. Arterial hypertension was encountered in 85 per cent of the bulbar cases, in 70 per cent of the spinal, and in 12 per cent of the nonparalytic polio. It was thought at first that hypoxia in the absence of visible cyanosis might be resulting in a compensatory elevation of the blood pressure. However, the persistence of hypertension in several instances for several days after continuous administration of oxygen by mask, intrathecally, and by tent, suggested that damage to the cardiovascular centers was probably the causative factor.

Lachmund[7] also described hypertension occurring in 22 of 208 patients with polio. His hypertensive patients were chiefly severely ill persons with bulbar poliomyelitis. Twelve of the 22 died.

Weinstein and Shelokov[8] found prolonged elevation of blood pressure in 7 per cent of 428 patients with acute polio. Hypoxia was thought to be the most probable cause of the hypertension in these patients.

Stimson has found among the cases he has seen and studied that in the early pure bulbar case, hypertension is a common symptom, and usually indicates a bad prognosis. It is postulated by Stimson and Pitts[49] that this initial hypertension seen in these cases is due to irritation of the vasomotor center by the initial invasion of the region by the virus—to be followed by vasomotor collapse and death when the virus invasion progresses to the point of destruction of these centers.

Fox et al.[20], in his article on bulbar polio, describes one group of cases which he calls the bulbar circulatory center group. This consists of patients in whom the signs of circulatory center failure are the most prominent feature. The usual findings were rapid, weak and irregular pulse, and marked hypertension. He also noted

that a fair per cent of these patients had an initial hypertension, especially marked in the diastolic readings. Terminally, there was complete circulatory failure. Often the heart sounds become inaudible before respiration ceased. In his series, 6 per cent of the bulbar cases were classified in this group, and in this group the mortality was the highest—92 per cent.

The regions of the central-nervous-system for which the virus has major predilection, as histologically determined, are the anterior horn cells, the cells of the motor nuclei of the brain stem, and the pre-ambital cells of the premotor cortex. Areas which are less frequently attacked include the spinal and bulbar internuncials, including the reticular substance, the visceromotor nuclei of the vagi and other ganglion cells, both centrally and peripherally.

Baker and Matzke have studied extensively the histopathology of poliomyelitis. A detailed histologic study of the entire medulla was undertaken in 80 cases of bulbar polio, and the pathologic alterations were correlated with the clinical symptoms.

In man the respiratory center appears to be localized to the small cells of the ventrolateral reticular formation. This is a paired center situated primarily between the descending root of the fifth cranial nerve and the inferior olivary nucleus. It extends from the superior pole of the twelfth nucleus to the bulbo-pontile junction. Focal areas of inflammatory necrosis destroying primarily these centers in poliomyelitis invariably result in clinical evidence of failure of central respiratory control, namely, irregular rhythm and depth of respiration with a tendency to shallowness and prolonged intervals between inspirations. These symptoms progress to periods of apnea and, finally, cessation of respiration. From their studies it appears that adequate allowance has been made for the protection of these centers. At least 31 per cent of these small reticular cells may be destroyed without precipitating clinically detectable alterations in respiration.

In 1939 Wang and Ranson, by stimulation experiments on cats, elicited strong pressor responses from the dorsomedial reticular substance at the level of the inferior fovea and the inferior olivary nucleus. Depressor responses were more diffuse than the pressor response and were located in the dorsolateral and ventromedial reticular substance. The observations of Baker and Matzke on man tend, in part, to substantiate these observations.

It also appears from their studies that the medullary vasomotor center in man is restricted to the large reticular cells of the ventromedial reticular substance. Bilateral focal necrosis situated just lateral to the midline, and destroying these cells, invariably produces clinical symptoms ascribable to the failure of central control of the circulation. The pulse becomes very rapid, often irregular, and at times difficult to palpate. The blood pressure may be elevated or may have a downward trend and become unobtainable. The pulse is low. Terminally the patient goes into shock with cyanosis, cold skin and hyperthermia. The cells of this nuclear group are very large, measuring 50 microns in diameter, and number about 100,000 on each side. Because of their relatively smaller number (as compared with respiratory cells), destruction of a minimum of 19 per cent of these large cells will precipitate vasomotor symptoms.

Invariably, when the course of the bulbar poliomyelitis was fulminating, with death within a short period, both the medial and the lateral cell groups were extensively destroyed. The course of the illness seemed to be much longer and less stormy when the lateral cell group, or the respiratory centers alone, were involved.

On the basis of the predominant functional injury to the cell groups in the medulla, it is possible to divide bulbar polio symptomatically into four types: (1) involvement of the nucleus ambiguous—resulting in dysphagia and dysarthria, (2) involvement of the small reticular cells situated in the ventrolateral reticular area and regulating respiratory function—resulting in irregular rhythm and depth of respiration, periods of apnea, and finally cessation of respiration, (3) involvement of the large reticular cells situated in the ventromedial reticular area and regulating circulation—resulting in clinical symptoms of vasomotor disturbance, (4) damage to both the large and small reticular cells, which, if extensive, results in rapid death.

Despite the general feeling[19 (2)] that hypoxia is not a primary factor in the sequela of poliomyelitis, it is clear that in the presence of virus-induced changes in nervous tissue, the additional burden of generalized hypoxia or local vascular changes in edema, may produce further impairment of the elements already involved and cause extension of damage to others which have previously escaped injury. Thus it is obvious that attention must be paid to these

nonspecific factors in the clinical management of the patient with poliomyelitis.

SUMMARY AND CONCLUSIONS

Hypertension is a phase of polio on which there is still much to be learned. In this paper has been presented a survey of the current knowledge about the causes of this hypertension—the direct effect of virus invasion of medullary centers, the effects produced by edema and stasis in the involved areas, and the part played by anoxia, resulting either from involvement of vital centers in the medulla or from peripheral muscular paralysis of the respiratory system (musculature).

Clinical and pathological findings in cases of polio where hypertension was present for any appreciable length of time have been presented.

When of a severe and protracted nature, this hypertension appears almost always to be of central nervous system origin. Good correlation has been found between the histological changes in the medulla and death from circulatory failure, respiratory collapse, or a combination of these. Transient hypertension on an anoxic basis, or secondary to an "alarm reaction" also occurs in polio as in many other conditions where one or more of these factors may be present. The incidence of this complication of polio does not seem significantly influenced by the patient's age or sex, although bulbar polio is somewhat more frequent in young males. Hypertension occurs in both mild and severe cases of polio, but is considerably more frequent in the latter.

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TREATMENT OF ULCERATIVE COLITIS IN CHILDREN. (Nordisk Medicin, Stockholm, 47: 13, Jan. 4, 1952). Bastman-Heiskanen says that ulcerative colitis may be regarded as a "psychicneuro-enzymatic-infectious" complex in which now one, now another, component dominates the picture. Psychiatric examination with a view to possible psychotherapy should precede somatic treatment In the acute phase, when bacterial activity seems to predominate, chloramphenicol is effective; in the more chronic cases, in which irritation in the vegetative system seems to be most conspicuous, administration of belladonna and phenobarbital gives good results. Surgical treatment of ulcerative colitis in children is not advised. Six cases of ulcerative colitis of from two months' to eight years' duration in children aged from 5 to 15 years are reported. In three cases there were more or less marked conflict situations in the home. In one case use of belladonna and phenobarbital resulted in noticeable improvement; in four out of five patients treated with chloramphenicol, partly alone, partly in combination with belladonna and phenobarbital, the results were good. Lysozyme determination in the feces, made in three of the cases, showed greatly increased lysozyme values; clinical improvement began with treatment with lauryl sulphate in one case, but no significant reduction in the lysozyme content was demonstrable. Early improvement occurred in a case in which ileostomy was done, but it was succeeded by spread of the ulcerative process to the ileum and fatal outcome. Attention is called to the periodicity of the disorder and the tendency to spontaneous recovery, believed to be especially marked during puberty.—*Journal A.M.A.*

PEDIATRICS AT THE TURN OF THE CENTURY

From time to time the Archives, which was the first Children's Journal in the English language, will reprint contributions by the pioneers of the specialty over fifty years ago. It is believed that our readers will be interested in reviewing such early pediatric thought.

CHRONIC CONSTIPATION IN CHILDREN*

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Judging by the construction of the infantile intestines—their great length, the thinness and feebleness of their musculature, etc. —nature seems to have intended that infants as well as older children should be more or less constipated. Indeed, the popular belief that healthy children are usually constipated is often corroborated by actual observation. Not infrequently, however, obstinate constipation gives rise to a number of disagreeable symptoms (flatulence, anorexia, headache, restlessness, sometimes convulsions, proctitis, anal fissure, prolapse of the rectum, hemorrhoids, etc.) requiring active treatment, a task often difficult to cope with in view of the uncertainty of the etiological factor of the underlying disease.

The causes of habitual constipation are very numerous. Aside from the cases resulting from gross abnormal anatomical relations or diseases, such as the different varieties of atresia intestini, recti, or ani; tumors, congenital dilatation with hypertrophy of the colon; hypertrophy of the valvulæ conniventes; hypertrophy of the socalled rectal valve; inflammatory adhesions; congenital displacements, etc.—which will not be discussed here—constipation is ordinarily caused by faulty diet, atony of the bowels, and constitutional disturbances.

Faulty Diet is responsible for a great many cases of constipation. This etiological factor is frequently potent also in infants, when the woman's milk contains too much or too little of one or more of the constituents of milk, or is insufficient in quantity. In artificially fed infants the cause of the constipation will probably

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be found in the insufficiency of fat consumed. In some children constipation is due, on the one hand, to too early and persistent feeding with amylacea, and on the other, to the consumption of food that does not stimulate peristalsis, such as an exclusive diet of milk, meat, eggs, etc., and no potatoes, bread, vegetables, etc.

Atony of the Intestines may be primary, congenital in nature, or secondary or acquired. The former variety can frequently be traced as an hereditary taint through several generations. Sometimes there is, in addition to the muscular insufficiency, also congenital weakness of the innervation of the intestines. The latter condition embraces also the form of atony usually associated with congenital diseases of the brain and spinal cord. Secondary or acquired intestinal atony is generally the result of repeated attacks of temporary constipation, gastro-intestinal indigestion with fermentation, enterospasm, arrest of peristalsis due to reflex irritation of the inhibitory nerves of the intestines, acute inflammatory processes of the intestinal canal with consecutive atrophy of the intestinal coats, constriction of the lumen of the bowels by temporary displacements (enteroptosis, hernia, etc.); habitual suppression of defecation or attention to it at irregular hours; enemas with large quantities of fluids, etc. All these etiological factors produce intestinal atony by directly or indirectly distending the lumen of the bowels and depriving the intestinal musculature of its resilience and tonicity. The latter condition is also apt to follow the abuse of antispasmodics, while drastic cathartics may lead to atony by mechanically thinning the intestinal coats.

In different chronic diseases associated with general debility, e.g., rachitis, and loss of flesh; in diseases of the nervous system, such as locomotor ataxia, myelitis, meningitis, etc., the sluggishness of the bowels forms merely a symptom of the principal disease. Habitual constipation is often met in diseases of the heart, profound anemia, etc., as a result of venous stasis of the abdominal organs; to the same cause is attributable also the constipation occurring in children who, through deformity or otherwise, are incapacitated to enjoy a sufficient amount of bodily exercise.

Treatment of obstinate constipation in infancy and childhood resolves itself, firstly, in arresting the causes instrumental in the production of the disease; secondly, in the removal of the damage done during the continuance of the constipation—not quite as easy

a task as some authors wish us to believe. Indeed, a good number of cases of chronic constipation are never cured, no matter what therapeutic means are being employed. Preventive measures are, therefore, to be recommended early and carried out with precision.

It is of primary importance to train the child to have a movement regularly every day. "Even in infants only a few months old proper habits are often easily formed if the child is put upon the chamber or chair invariably at the same hour." The first few days it may require local stimulation to defecation, e.g., introduction into the rectum of a small oiled syringe-tip. Similar means should be employed also with older children and particularly school children who are very apt to suppress nature's impulse to empty the bowels.

Two main factors are instrumental in the expulsion of the rectal contents, contraction of the abdominal muscles and the diaphragm, and separation or relaxation of the gluteal group of muscles. "When the child is old enough to be placed upon a commode chair it is often in such a position as to make defecation very difficult indeed. Its trunk is bolt upright, the feet dangle from a seat which is too high, and the expulsive power of the abdominal muscles is reduced to a minimum. Besides, the aperture in the seat is so wide that no support is given to the tubera ischii, the gluteal masses are crowded together instead of separated and the descent of the floor of the perineum is much hindered." This impediment to defecation may be obviated by substituting a low seat on a nursery chair or closet or small vessel for the high one previously used. The child is thus enabled to accomplish this act in a squatting posture which is most favorable to the thorough emptying of the rectum.

Correction of diet is, of course, very valuable for the prevention of habitual constipation, but does not always remedy the trouble. This is particularly true of cases of very long standing, since here we are dealing with secondary atony following prolonged distension and enfeeblement of the intestines. With the introduction of the recent methods of percentage feeding and the employment of "top milk" as a base, and barley- or oatmeal-water as a diluent, the number of cases of obstinate constipation among bottle-fed infants, due solely to faulty feeding, has perceptibly diminished. Hence, the indication of these methods of feeding also as a corrective of

constipation. In breast-fed infants attention should be directed to the improvement of the general nutrition of the mother or wet nurse. Frequently, however, it is almost impossible to regulate the quantity of fat in breast-milk. In this event the deficiency in fat may be supplied by administering to the infant, just before nursing, a teaspoonful or two of sweet cream. The addition of cream, malt preparations, buttermilk, honey, an extra supply of cooked or raw fruit and vegetables to the regular "mixed-diet" is invaluable as a corrective of constipation also in older children. A glass of cold water on an empty stomach and at night before retiring is often very useful.

Faithful compliance with the suggestions just made very often yields favorable results. In a certain percentage of cases, however, more active measures have to be resorted to and it then devolves upon the physician to select such therapeutic means as will not affect the general well-being of the patient. This indication can most appropriately be met by the simultaneous employment of a combination of the socalled physico-chemical procedures, consisting of massage, oil enemas and hydrotherapy, and occasionally, also, electricity. This treatment is more advantageously carried out in the evening, before the patient goes to sleep. The child is placed on a hard couch or mattress with head and thorax raised and legs sharply flexed at the knee joint and somewhat rotated outward. The attendant stands on the left side of the patient. The manipulations are begun at the fossa iliaca sinistra,* where the sigmoid flexure is situated and is frequently found to be a halting place for hardened feces. With the tips of the fingers of one hand (in older children both hands may be used, one hand being placed upon the other) the attendant makes gentle circular movements along this portion of the colon and at the same time exerts upon it considerable pressure downwards towards the rectum. Without changing these movements the attendant slowly ascends as far as the splenic flexure. From here he gradually returns to the sigmoid. He now begins a new tour going as far as the hepatic flexure,

*Most authors recommend to begin the massage over the cecum, but I fully agree with the following drastic remarks of Thure Brandt (Dr. E. Ekgren's Manual on Massage): "In attempting to express the contents of a long sausage it will prove quite difficult to do so by beginning to push the entire contents from the proximal to the distal end; on the other hand, if begun with the distal, open end, the sausage can be emptied piecemeal without any difficulty whatever." The same reasoning figuratively applies to the intestines.

and after gradually returning to the starting point he makes his final trip reaching the cecum and, in the manner just outlined, returns again to the fossa iliaca sinistra. These manipulations should be followed by rhythmical vibratory strokes over the entire abdomen, interrupted by a few pressure movements against the spinal column in the epigastric region. The treatment should last from six to twelve minutes.

Instead of trying the massage, oil enemas, and hydrotherapy separately, it is certainly preferable to employ these three procedures—the anticostive triad—simultaneously, as they do not interfere with one another, but, on the contrary, are destined to supplement one another in their beneficial effect. Thus, after completing the massage the little patient is turned upon his left side, and by means of a piston syringe a half an ounce or more of oil is gently injected into the rectum and allowed to remain there. This is followed by the application around the abdomen of a Priessnitz compress, which should be left in place until the next morning. It will almost invariably be found that the patient's bowels will act either during, or soon after the treatment or, at any rate, not later than the following morning. A three or four weeks' course of treatment will usually suffice to establish regularity of the bowels provided the preventive measures suggested before are strictly adhered to. In some, very protracted cases of constipation these procedures may be supplemented by the application of the galvanic or faradic current. One electrode is passed successively over different portions of the abdominal wall, and the other electrode is placed upon any other part of the body.

Proctologists frequently advocate divulsion of the sphincter ani as a sure cure of habitual constipation. I am not inclined to be quite as enthusiastic over it, except in cases of constipation due to rectal disease, as, for example, fissura ani, rectospasmus, etc.

Finally, there is a class of cases of chronic constipation which resists all forms of treatment as regards a permanent cure, but may be considerably improved by alternately resorting to the therapeutic measures already enumerated as well as to drugs. In the selection of an evacuant the physician must be guided by the etiological factors and the individual peculiarities of the case in question. The indiscriminate use of antispasmodics as well as the ever ready, "soothing" laxatives is to be strongly deprecated.

"That only is a good purgative which will produce without discomfort (colic, tenesmus, nausea), a soft, not a watery, abundant evacuation." Effective and comparatively harmless are the following remedies: Soap and glycerin suppositories, medicated cocoa butter suppositories (with aloin and belladonna in spastic, or nux vomica in atonic constipation), enemas with small quantities of glycerin or larger quantities of soap-water; internally magnesia usta, magnesia and rhubarb, compound licorice powder, castor-oil, extract of cascara sagrada, calomel followed by a mild saline aperient, and, in older children, the standard mineral salts or waters. Of the more recently recommended cathartics I found exodin (diacetyl-rufigallic-acid-tetramethyl-ether) to act splendidly. It is tasteless and, therefore, readily taken by the little patients either in apple sauce, syrup of acacia or in tablet form. In from three to ten grains it produces a soft movement without any gastro-intestinal disturbance.

Whatever the method of treatment employed, the establishment of a habit of regular stools should at all times be our chief aim.

CAT SCRATCH FEVER. (Union Médicale du Canada, Montreal, 81: 123, Feb. 1952.) Acute benign infectious lymphoreticulosis is not always caused by contact with cats; infection sometimes occurs as a result of brier scratches, superficial injuries from splinters, metal fragments, and the like, and may even be contracted through the eyes, mouth, or pharynx. The disease may be troublesome, but its evolution is favorable, ending in cure. At the outset, glandular tuberculosis or Hodgkin's disease may be suspected; later, the condition may be mistaken for venereal lymphogranuloma. Biopsy reports are not always conclusive, and sometimes they tend to confirm the diagnosis of Hodgkin's disease. Negative reactions to various tests, notably to Frei antigen and tuberculin, will eliminate other possible conditions, and a positive intradermal reaction to the specfic antigen of lymphoreticulosis will establish the diagnosis. The prognosis is good, but the course of the disease is variable. There may be spontaneous resolution or more or less prolonged suppuration. Terramycin accelerates healing and is apparently better than chloramphenicol (chloromycetin) or aureomycin.—*Journal A.M.A.*

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